Failure to Cure *Mycobacterium gordonae* Peritonitis Associated with Continuous Ambulatory Peritoneal Dialysis

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Nontuberculous mycobacteria are increasingly recognized as important pathogens in peritonitis associated with continuous ambulatory peritoneal dialysis (CAPD). *Mycobacterium gordonae* rarely causes human infection and is the least likely mycobacterium to produce clinical infection in CAPD patients. We describe a patient with persistent *M. gordonae* peritonitis acquired while undergoing CAPD. During 18 months of treatment, clinical improvement occurred but a microbiological cure could not be achieved. Principles of therapy for mycobacterial peritonitis developing during CAPD are reviewed, and potential explanations for our patient’s failure to respond to therapy are discussed.

Infection is the most common medical complication of continuous ambulatory peritoneal dialysis (CAPD) [1]. Frequent sites of infection include the dialysis catheter exit site, the subcutaneous catheter tunnel, and the peritoneum [2]. Peritonitis is the most serious infection and frequently necessitates at least temporary cessation of CAPD.

CAPD peritonitis is usually caused by a single organism, and this is commonly an aerobic gram-positive bacterium [1]. However, numerous other organisms cause peritonitis in CAPD patients, including mycobacteria. Although nontuberculous mycobacteria account for <3% of cases of CAPD-related peritonitis, infection with these organisms often provides formidable diagnostic and therapeutic challenges [1]. We describe a patient with recalcitrant *Mycobacterium gordonae* peritonitis acquired during CAPD.

**Case Report**

A 39-year-old woman with end-stage renal disease secondary to chronic glomerulonephritis underwent CAPD for 5 years, and her dialysis effluent became cloudy in January 1994. She had several prior episodes of bacterial peritonitis while undergoing CAPD, as well as fungal peritonitis due to *Candida parapsilosis* in June 1993, which required removal of a Tenckoff catheter. Inadvertent bowel perforation during replacement of the Tenckoff catheter necessitated surgical repair in July 1993.

The patient denied fever, chills, and abdominal pain despite the cloudy effluent. Findings of a physical examination were normal. Analysis of the dialysis effluent revealed a WBC count of 990/mm³, with 83% neutrophils, 15% macrophages, 1% lymphocytes, and 1% monocytes (table 1). Cultures for bacteria and fungi were negative, and no acid-fast bacilli (AFB) were seen on a Kinyoun-stained concentrated smear.

The cloudy effluent cleared with abdominal lavage. Vancomycin therapy was discontinued after one dose because of negative culture results. Three weeks later, a culture of the fluid yielded yellow-pigmented colonies. The scotochromogenic isolate was identified as *M. gordonae*. No treatment was instituted at that time.

In February 1994 the dialysate WBC count was 141/mm³, with 92% neutrophils, 7% macrophages, and 1% monocytes. No organisms were seen on gram stain, and no AFB were seen on a concentrated smear. Four weeks later, an AFB culture again yielded yellow-pigmented colonies that were identified as *M. gordonae*, and antimycobacterial therapy was initiated with isoniazid (300 mg/d), rifampin (600 mg/d), and pyrazinamide (1,000 mg/d).

In March 1994 the peritoneal dialysate WBC count was 19/mm³, with 100% neutrophils. AFB were evident on direct examination of a Kinyoun-stained concentrated smear. One week later, the patient presented to the renal clinic because of a 4-day history of fever, abdominal pain, and a palpable, tender right-abdominal mass just lateral to the umbilicus.

A CT scan revealed an abdominal-wall fluid collection with multiple loculations. At laparotomy, the abscess was noted to communicate with the abdominal cavity, and widespread adhesions were visualized. Several pockets of purulent fluid were drained, and a large amount of necrotic fat, muscle, and fascia was debrided. Complete surgical drainage was prevented by the extensive adhesions.

Peritoneal fluid obtained at surgery had a WBC count of 580/mm³, with 78% neutrophils, 10% monocytes, 10% lymphocytes, and 2% eosinophils. Gram staining and bacterial and fungal cultures demonstrated no organisms, but numerous AFB were seen on a concentrated smear. Similarly, abdominal wall fat contained large numbers of AFB. The peritoneal dialysis catheter was removed, maintenance hemodialysis was initiated, and the patient’s antimycobacterial therapy was changed to...
administration of ethambutol (1,000 mg/d), rifampin (600 mg/d), and intravenous amikacin (80 mg after each hemodialysis treatment).

The patient’s abdominal pain abated after surgery. The peritoneal isolate recovered in February was confirmed by the National Jewish Center for Immunology and Respiratory Medicine to be *M. gordonae*. In addition, the peritoneal fluid cultures performed in March 1994 also yielded *M. gordonae*.

Drug susceptibility testing performed with BACTEC methodology (Becton Dickinson, Sparks, MD) demonstrated susceptibility to ciprofloxacin, rifabutin, and ethambutol; moderate susceptibility to ciprofloxacin and rifampin; moderate resistance to isoniazid and amikacin; and resistance to pyrazinamide, imipenem, and streptomycin. Susceptibility to clarithromycin could not be determined because this organism does not grow at a pH of 7.4, which is necessary for clarithromycin testing. Susceptibility to cyclosporine, ethionamide, and kanamycin was not tested.

The patient received ethambutol (1,000 mg/d), rifampin (600 mg/d), and amikacin (80 mg post-dialysis) until August 1994, when concerns about potential ototoxicity prompted withdrawal of amikacin and initiation of administration of clarithromycin (500 mg/d). A subsequent CT scan showed two large, loculated fluid collections in the left flank and a smaller collection in the mid-abdomen, adjacent to the abdominal wall. A diagnostic aspirate of this latter collection was obtained, revealing many AFB on a concentrated smear, but cultures of this specimen yielded no growth.

Percutaneous drainage of the left flank collections was performed in October 1994, and again numerous AFB were seen on a concentrated smear. A follow-up CT scan in March 1995 revealed persistent abdominal fluid collections, a smear showed AFB, but mycobacteria did not grow in culture.

The patient was treated with ethambutol (1,000 mg/d), clarithromycin (500 mg/d), and rifampin (600 mg/d) to complete 18 months of therapy. Intermittent fevers persisted for 18 months. At the time this article was written, the patient had no abdominal pain, but she had early satiety, malnutrition with a low serum albumin concentration (3.2 g/dL), and a 15-kg weight loss she had never regained.

### Table 1. Results of mycobacterial smears and cultures for a patient with peritonitis associated with CAPD.

<table>
<thead>
<tr>
<th>Specimen collection date</th>
<th>Type of specimen</th>
<th>WBCs (no./mm³)</th>
<th>Presence of AFB in concentrated smear</th>
<th>Isolate from final culture (date)</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 Jan 1994</td>
<td>CAPD effluent</td>
<td>990</td>
<td>No</td>
<td><em>M. gordonae</em> (17 Feb 1994)</td>
</tr>
<tr>
<td>18 Feb 1994</td>
<td>CAPD effluent</td>
<td>141</td>
<td>No</td>
<td><em>M. gordonae</em> (16 Mar 1994)</td>
</tr>
<tr>
<td>5 Apr 1994</td>
<td>Abdominal-wall tissue</td>
<td>. . .</td>
<td>Yes</td>
<td><em>M. gordonae</em> (27 Apr 1994)</td>
</tr>
<tr>
<td>6 Apr 1994</td>
<td>Peritoneal fluid</td>
<td>580</td>
<td>Yes</td>
<td><em>M. gordonae</em> (4 May 1994)</td>
</tr>
<tr>
<td>8 Apr 1994</td>
<td>Abdominal-wall tissue</td>
<td>. . .</td>
<td>Yes</td>
<td><em>M. gordonae</em> (19 May 1994)</td>
</tr>
<tr>
<td>5 Aug 1994</td>
<td>Peritoneal fluid</td>
<td>304</td>
<td>Yes</td>
<td>No growth</td>
</tr>
<tr>
<td>5 Oct 1994</td>
<td>Peritoneal fluid</td>
<td>225</td>
<td>Yes</td>
<td>No growth</td>
</tr>
<tr>
<td>10 Mar 1995</td>
<td>Peritoneal fluid</td>
<td>150</td>
<td>Yes</td>
<td>No growth</td>
</tr>
</tbody>
</table>

NOTE. AFB = acid-fast bacilli; CAPD = continuous ambulatory peritoneal dialysis.

### Discussion

The association between end-stage renal disease and increased susceptibility to tuberculous and nontuberculous mycobacterial disease has been well documented [3-5]. These nontuberculous mycobacterial infections have predominantly involved extrapulmonary sites, including the peritoneum [3].

The most common etiologic organisms are representatives of Runyon group IV (rapid growers), such as *Mycobacterium chelonae* and *Mycobacterium fortuitum* [6]. *M. gordonae* (a member of Runyon group II) is considered the least pathogenic mycobacterium and has rarely been implicated in disease [7]. In fact, only one other case of CAPD-related peritonitis secondary to this organism has been reported. In the case described by London and colleagues [8], removal of the Tenckhoff catheter alone failed to eliminate the infection. Antimycobacterial therapy with isoniazid, rifampin, ethambutol, and postdialysis amikacin for 2 months, followed by isoniazid, rifampin, ethambutol, and pyrazinamide for 4 months, resulted in clinical cure [8].

Like any of the other nontuberculous mycobacteria, *M. gordonae* is ubiquitous and has been isolated from numerous environmental sources, including house and hospital tap water, soil, and raw milk [9]. Since *M. gordonae* is so widespread in the environment and is typically innocuous, discerning true infection from colonization or culture contamination is a clinical challenge.

In our case, peritoneal infection with *M. gordonae* was well documented on the basis of the criteria outlined by Weinberger and colleagues [7]. The organism was observed on a Kinyoun-stained smear and isolated in cultures of multiple specimens of peritoneal fluid. In addition, necrotic fat from the abdominal wall contained the organism, as revealed by microscopy and by culture.

Though initially the clinical course of the patient was inconsistent with peritoneal infection, ultimately she had classic signs and symptoms of peritonitis, as well as findings consistent with a contiguous abdominal-wall abscess. However, despite removal of the peritoneal dialysis catheter, significant surgical debridement, abdominal percutaneous drainage, and appro-
appropriate, susceptibility test–guided antimicrobial therapy for 18 months, the patient’s infection has not been clinically cured since she is still malnourished and has been unable to regain a 15-kg weight loss.

Although the inability to grow M. gordonae in culture after April 1994 suggests a microbiological cure, the progressive abdominal fluid collections with elevated WBC counts and with large numbers of AFB on smears, even after 1 year of antibiotic therapy, suggest a possible microbiological failure.

Several factors impacted the response of our patient to therapeutic interventions. It is well known that patients with end-stage renal disease have impaired cell-mediated immunity [3]. The prior episode of fungal peritonitis and the current infection with M. gordonae suggest impaired delayed hypersensitivity in this patient. In addition, for a variety of reasons, including the initial absence of focal clinical symptoms, the general lack of pathogenicity of M. gordonae, the lengthy culture recovery time for the organism, and the history of vascular-access problems in this patient, the dialysis catheter was not removed until 2 months after the initial isolate was recovered.

The presence of this foreign body may have facilitated progressive peritoneal seeding, although successful therapy without catheter removal may occur [1, 10–12]. In addition, because the patient had such extensive abdominal adhesions, and since the mycobacterial infection was widely spread throughout the abdominal cavity, complete surgical exploration and debridement were impossible. This may have resulted in residual foci of infection that continued to shed organisms within the peritoneum.

Finally, it is possible that the antibiotics utilized on the basis of in vitro susceptibilities lacked similar in vivo effectiveness [13–15]. Current methodologies for testing the drug susceptibility of nontuberculous mycobacteria are based upon models developed for traditional antituberculous agents [15]. However, these methods have not always yielded a positive correlation between in vitro susceptibility and clinical response [14]. As the incidence of nontuberculous mycobacterial infections rises in this era of increasing immunosuppression, and as resistant organisms inevitably emerge, the development of laboratory testing that correlates with clinical efficacy is vital.

In contrast with the patient described by London and colleagues [8], our patient failed to respond to medical and surgical therapy, and clinical cure and (probably) microbiological cure were not achieved. Therapy was delayed because of a lack of awareness of the pathogenicity of M. gordonae and the late availability of results of cultures and drug-susceptibility tests. However, if therapy had been initiated earlier, it is possible that a cure could have been achieved. M. gordonae should now be included in the differential diagnosis of the etiology of peritonitis in patients undergoing CAPD.

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References